Original Article

Role of sucralfatein acid peptic disease: A study of 50 cases

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Abstract

Introduction: Peptic ulcer treatment has for many years been Concentrated on reduction of intraluminal acidity. The efficacy of antacid agents in the treatment of gastric ulcer is, however, uncertain. Oxethazine, an H2-receptor antagonist with high acid reducing capacity, has been demonstrated in a few controlled studies to have a statistically better healing efficacy in gastric ulcer than placebo. The development of agents acting on the gastric mucosal barrier level, and having cytoprotective properties has provided a new method of combating peptic ulcer disease. Sucralfate, a basic aluminum salt of sucrose octasulphate, is one of the new cytoprotective agents and has in previous studies given healing rates comparable with those given by ranitidine in the treatment of both duodenal and gastric ulcer. **Aims and Objective:** The main purpose of this study the healing rates and symptom relief in patients with acute peptic ulcer treated with Sucralfate and Oxethazine Containing Antacids. **Methodology:** This was case series of 50 known cases of Peptic Ulcer disease 25 using Sucralfate (Group A) for treatment and 25 Oxethazine Containing Antacids for the (Group B) treatment. All the patients who have given informed written consent were included into study except patients having hypophosphatemia severe renal defect, Pregnancy, Any severe illness. Graph Pad Prism software was used to calculate statistical significance. **Result:** That the main risk factors were Mean age54±10.11 and 55.5±9.6, female sex 12/13 and 11/14, Smoker 42% and 43%, NSAIDS 24% and 23%, Alcohol 64%, 67%, Coffee 80%, 88% were present in both the Group i.e. Sucralfate (Group A) and Oxethazine Group (Group B) and the risk factors were equally distributed in both the groups as (p.0.05,ns).At the beginning of the treatment in Group A and Group B was14.1±6.1mm and 13±5.5mm respectively so there was not statistical difference in between them (p>0.05,t=0.89,df=48,ns). At 8 weeks also the size were 8.8±4.4mm and 11±5.4mm mm and there was not statistical difference in between them (P>0.05,t=1.85,df=48,ns). 12 weeks the mean size was 4.1±1.2mm and 9.5±5.4mm there was significant statistical difference in between them (P<0.001, t=6.5, df=48,hs). 16 weeks the mean 2.2±.82mm and 7.5± 3.4mm, highly significant statistical difference (*P<0.005,t=5.5,df=48,hs) respectively in Group A and Group B. **Conclusion:** Sucralfate has excellent role in the ulcer healing over the Oxethazine group so; it should be used in the treatment of peptic ulcer where ever it is possible.
Key Words: Sucralfate, Oxethazine, NSAIDs

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INTRODUCTION

Peptic ulcer treatment has for many years been Concentrated on reduction of intraluminal acidity. The efficacy of antacid agents in the treatment of gastric ulcer is, however, uncertain.[1] Oxethazine, and H2-receptor antagonist with high acid reducing capacity, has been demonstrated in a fewcontrolled studies to have a statistically better healing efficacy in gastric ulcer than placebo.[2-3]The development of agents acting on the gastric mucosal barrier level, and having cytoprotectiveproperties has provided a new method of combatingpeptic ulcer disease. Sucralfate, a basic aluminum salt of sucroseoctasulphate, is one of the new cytoprotective agents and has in previous studies given healing rates comparable with those given by cimetidine in the treatment of both duodenal and gastric ulcer.[4-7] The most important aspect is to treat the underlying cause Antacids, H**2**-blockers, sucralfate and proton pump inhibitors have been tried, either alone or in combination[8, 9]**.** It is a complex. Salt of sucrose sulphate and aluminium hydroxide, which is poorly soluble in water and

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minimally in dilute acids and alkalies. When dissolved in stomach contents, after releasing aluminum salt it becomes strongly negative and combines withmucin to form a viscous suspension that binds with normal as well as defective mucosa. It binds pepsin but lacks anti-ulcer efficacy[10, 11].A combination of different actions enables sucralfate to prevent mucosal injury; these are its antiseptic effect, acting as a physical barrier, increasing the production, viscosity, hypophoacidity and aluminum, carbohydrate content of mucosa making it more acid resistant. It also promotes prostaglandin mediated and independent bicarbonate output from gastric and duodenal mucosa its effect on tissue growth regeneration and repair is also contributory [10-13]. Sucralfate has been reported as a safe drug during last 10 years of its use[14-15]. No systemic toxicity, teratogenicity or tumour producing effect has been reported, except hypophosphatemia and aluminum intoxication in patients with renal defect.[16-17]

AIMS AND OBJECTIVE

The main purpose of this study was to compare the healing rates and symptom relief in patients with acute peptic ulcer treated with Sucralfate and Oxethazine.

METHODOLOGY

This was case series of 50 known cases of Peptic Ulcer disease 25 using Sucralfate (Group A) for treatment and 25 Oxethazine Containing Antacids for the (Group B) treatment. All the patients who have given informed written consent were included into study except patients having hypophosphatemia severe renal defect, Pregnancy, Any severe illness. Graph Pad Prism software was used to calculate statistical significance.

RESULTS

Table 1: Distribution of the Patients as per the associated Risk-Factors

Risk Factors	Sucralfate (n=25)	Oxethazine(n=25)	p-Value
Mean age ± SD.	54±10.11	55.5±9.6	p>0.05, ns.
Sex –Male/Female	12/13	11/14	p>0.05, ns.
Smokers	(42%)11	(43%)12	p>0.05, ns.
NSAIDS	(24%)6	(23%)6	p>0.05, ns.
Alcohol	(64%)16	(67%)3	p>0.05, ns.
Coffee.	(80%)20	(88%)22	p>0.05, ns.

'ns-not significant, hs-highly significant'.

From **Table 1:** It shows that the main risk factors were Mean age54±10.11 and 55.5±9.6, female sex 12/13 and 11/14, Smoker 42% and 43%, NSAIDS 24% and 23%, Alcohol 64%,67%,Coffee 80%, 88% were present in both the Group i.e. Sucralfate (Group A) and Oxethazine Group (Group B) and the risk factors were equally distributed in both the groups as (p>0.05,ns)

Table 2: Distribution of the patients as per Average Size of Ulcer in Group A and Group B

Time since treatment (in Weeks)	Average Size of Ulcer in Group A (Mean±SD)	Average Size of Ulcer in Group B (Mean±SD)	p-value (unpaired t-test)
At the Starting (0 weeks)	14.1±6.1mm	13±5.5mm	p>0.05,t=0.89,df=48,ns
8 weeks	8.8±4.4mm	11±5.4mm	P>0.05,t=1.85,df=48,ns
12 weeks	4.1±1.2mm	9.5±5.4mm	*P<0.001,t=6.5,df=48,hs
16 weeks	2.2±.82mm	7.5± 3.4mm	*P<0.005,t=5.5,df=48,hs

' ns-not significant, hs-highly significant'.

From **Table2:** It is clear that At the beginning of the treatment in Group A and Group B was14.1±6.1mm and 13±5.5mm respectively so there was not statistical difference in between them (p>0.05,t=0.89,df=48,ns). At 8 weeks also the size were 8.8±4.4mm and 11±5.4mm mm and there was not statistical difference in between them (P>0.05,t=1.85,df=48,ns). 12 weeks the mean size was 4.1±1.2mm and 9.5±5.4mm there was significant statistical difference in between them (P<0.001, t=6.5, df=48, hs). 16 weeks the mean 2.2±.82mm and 7.5± 3.4mm, highly significant statistical difference (*P<0.005, t=5.5, df=48, hs) respectively in Group A and Group B.

DISCUSSION

Mucosal lesions in the upper gastrointestinal tract are believed to be due to an imbalance between mechanisms maintaining the integrity of the mucosa and synthesis and release of prostaglandins. Cytoprotection has been defined as the ability of drugs to protect the gastric mucosa from necrotic damage by a variety of aggressive agents like alcohol, boiling water, acid, and over distension. Sucralfate has long been thought to heal ulcers

by forming a protective barrier over eroded mucosa. This barrier is impermeable to acid; binds bile salts and inhibits pepsin activity.'[18]recent investigations have shown that sucralfate also possesses cytoprotective properties, probably via stimulation of the local synthesis and release of prostaglandins. Mucosal blood flow, epithelial cell renewal and the formation of mucus is thereby enhanced. This, together with the absence of known interactions, extremely low toxicity and side effect makes this drug highly interesting in the treatment of gastric ulcer. In our study we found that the main risk factors were Mean age54±10.11 and 55.5±9.6, female sex 12/13 and 11/14, Smoker 42% and 43%, NSAIDS 24% and 23%,Alcohol 64%,67%,Coffee 80%, 88% were present in both the Group i.e. Sucralfate (Group A) and Oxethazine (Group B) and the risk factors were equally distributed in both the groups as (p.0.05,ns) as there was no any statistical differences in risk factors in these two groups so matching of the risk factors were perfect and whatever the significant effect was found in the treatment regimen was only because of the drugs. It is clear that At the beginning of the treatment in Group A and Group B was14.1±6.1mm and 13±5.5mm respectively so there was not statistical difference in between them (p>0.05,t=0.89,df=48,ns). At 8 weeks also the size were 8.8±4.4mm and 11±5.4mm mm and there was not statistical difference in between them (P>0.05,t=1.85,df=48,ns). 12 weeks the mean size was 4.1±1.2mm and 9.5±5.4mm there was significant statistical difference in between them (P<0.001, t=6.5, df=48, hs). 16 weeks the mean 2.2±.82mm and 7.5± 3.4mm, highly significant statistical difference (*P<0.005, t=5.5, df=48, hs) respectively in Group A and Group B. This means that significantly sucralfate have excellent effect of ulcer healing property under Endoscopy as compared to cimetidine, even the size of the ulcers were also decreased in the ranitidine group but the decrease was not that much significant as compared to sucralfate.

CONCLUSION

Sucralfate has excellent role in the ulcer healing over the Oxethazine group so; it should be used in the treatment of peptic ulcer where ever it is possible.

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